

From the University Eye Clinic, Basel
(Dir.: Prof. Dr. F. Rintelen)

Horizontal Hemianopias

A Review Prompted by a Case of Bilateral Horizontal
Hemianopia Affecting the Upper Half of the Visual Field

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A Review Prompted by a Case of Bilateral Horizontal Hemianopia
Affecting the Upper Half of the Visual Field

By A. SCHÄUBLIN

I. General Observations

(1) Definition and Pathogenetic Principles

The term horizontal or altitudinal hemianopia is applied to defects of the visual field in which the horizontal meridian of the field corresponds either exactly or approximately to the dividing line between the unaffected and the blind portion of the visual field. In French the condition is referred to as *hémianopsie horizontale* or *hémianopsie altitudinale*, and in German as *horizontale Hemianopsie* or *Höhen-Hemianopsie*.

The disorder may affect the upper or the lower half of the field of vision in either one or both eyes (*hemianopsia horizontalis superior* or *inferior*, *monolateralis* or *bilateralis*). Where both eyes are involved, the defects may be symmetrical, although cases of crossed hemianopia also occur in which there is loss of vision in the upper half-field of one eye and in the lower half-field of the other eye (*Traquair*).

It would be as well, first of all, to make a critical examination of the term "horizontal hemianopia", which usually refers simply to the shape of the defect in the visual field, i.e. it sheds no light on the question as to the location of whatever pathological process it is that has provoked the condition, nor does it touch on the related problem of whether the noxa is a unilateral or a bilateral one. The only purpose served by the expression "horizontal hemianopia", in fact, is to convey an idea of the repercussions of the damage in question, which must be located somewhere in the course of either one or both visual pathways. In contrast to this is the strict definition of hemianopia as a loss of vision which usually involves both visual fields, which affects identical or symmetrical halves of the retina in at least ap-

proximately the same form and degree, and which is attributable to a single lesion (*Behr*).

To satisfy this definition, the lesion has to interfere with the visual pathway at some point from the optic chiasma upwards. If they are to be used correctly, the terms "monolateral" and "bilateral hemianopia", too, should be employed in accordance with these rules. As a unilateral defect of the visual field, monolateral hemianopia must also be due to a lesion whose location is such that, in point of fact, one would expect it to produce either homonymous or heteronymous loss of vision in both visual fields. Of such cases that have been reported (quoted in *Lauber*), most have been accounted for as examples of higher-grade homonymous hemianopic asymmetries occurring in the presence of damage to the optic tract. What may also be regarded as a classic example of unilateral hemianopia is an isolated defect of a temporal demilune, i.e. of that portion in the monocular visual field which occurs as a non-paired entity in the binocular field.

According to its definition, bilateral hemianopia must be attributable to two causative lesions situated between the optic chiasma and the visual cortex.

In answer to the question as to where in the course of the visual pathway damage has to occur in order to produce defects in the upper or lower half of the visual field which do not extend beyond the horizontal meridian, it must be concluded that, in the case of unilateral loss of vision in either the upper or lower half of the field, the damage is located at some point anterior to the chiasma. On the other hand, if the hemianopia is bilateral, it must be assumed that a single pathological process is acting symmetrically on the chiasma or, alternatively, that there are two symmetrically located noxae present either anterior to the chiasma or somewhere in the course of the two higher visual pathways; the fact that, in this case, the two lesions would have to be present simultaneously, and to be of equal severity and extent, explains why bilateral horizontal hemianopia is such a rare condition. Finally, although it is theoretically conceivable that noxae differing in type or location on the one side as compared with the other could cause horizontal defects in the visual field, this is extremely unlikely.

If we compare these pathogenetic principles with the requirements which must of necessity be fulfilled in order that the condition may comply with the definition of hemianopia, it becomes obvious that

the type of visual-field defect normally referred to as unilateral horizontal hemianopia is in point of fact not a genuine form of hemianopia. Since in such cases the location of the damage is pre-chiasmal, and the damage can therefore affect vision in one eye only, it would be more correct to describe the disorder, for example, as "a complete or partial horizontal loss of vision".

Genuine horizontal hemianopia can be said to exist only in cases where it is due to damage affecting the optic chiasma or where two symmetrically situated lesions in the course of both higher visual pathways have resulted in bilateral homonymous quadrantic loss of vision. Since in the literature, however, the term "horizontal hemianopia" is generally used in its broader sense (i.e. solely by reference to the shape of the visual field), we shall continue to employ this term here, even in instances where – in view of what has just been said – it is actually incorrect to speak of horizontal hemianopia. We have nevertheless included these introductory remarks in an attempt to clarify the question of terminology.

(2) Location of Causative Lesions and Anatomical Observations

A study of the literature reveals that almost all structures of the visual pathway are liable to sustain damage which may give rise to horizontally bounded defects of the visual field. Such lesions may be located at any of the following sites: intra-ocularly; in the optic nerve, including particularly its intracanalicular and intracranial portions; in the optic chiasma; in the lateral geniculate body; and in either the optic radiation or the visual cortex.

If a lesion situated somewhere along the visual pathway is to produce a horizontal hemianopia, and if – from the shape of a defect in the visual field – we are to succeed, quite generally, in drawing diagnostic conclusions as regards the severity, extent, and location of the lesion, we must be able to proceed on the assumption that the visual pathway is built up in a strictly regular fashion, i.e. in such a way that the specific sectors of a cross-section through the visual pathway each have their own corresponding and precisely defined "spatial values" in the visual field. Nowadays we have a good deal of information on the architecture of the cells and fibres which constitute the anatomical substrate involved in the study of visual-field defects. Our knowledge is based, firstly, on microscopic studies carried out by older pathologists with reference to lesions of varying type and localisation in the visual pathway and, secondly, on experimental research

concerned with processes of degeneration, as undertaken by such authors as *Brouwer and Zeeman*, *Hoyt and Luis* in particular, as well as by *Polyak*. Where there are still uncertainties, it is upon the following two points that they are chiefly centred:

(a) The problem as to whether the crossed and uncrossed fibres in the optic tract run intermingled with one another or separately. If there is a large degree of spatial segregation between these fibres, this might account for the occurrence of highly asymmetrical homonymous or even unilateral hemianopias in the presence of damage to the optic tract.

(b) The significance of the synapses in the lateral geniculate body. This point, however, is only of secondary clinical importance, because it has no bearing on the principle that sectors of the visual field are projected on to the cross-sections of the visual pathway in conformity with a regular pattern.

Sufficient is now known about the architecture of the fibres and cells concerned to enable us to offer a theoretical explanation for a horizontal hemianopia at any point along the visual pathway.

Finally, another problem to be considered when discussing hemianopias is the question as to how the two main meridians of the visual field which pass through the fixation point, i.e. the vertical and the horizontal meridian, are represented in the visual pathway. The vertical meridian has no pre-chiasmal anatomical substrate. Since homonymous hemiretinae are represented in each of the higher visual pathways, this meridian is potentially present after the fibres have crossed; its actual presence, however, is invariably pathological. For the horizontal meridian, a multiple morphological correlate can be found: the horizontal meridian is – to some extent at least – directly visible as the temporal raphe of the retina and is also represented by the posterior horns of the lateral ventricle (which spatially separate the optic-radiation fibres belonging to the superior and inferior retinal quadrants) and, in the cortical region, by the calcarine fissure.

(3) *Incidence of Horizontal Hemianopia*

Horizontal hemianopia is not a common type of visual-field defect. The figures available on such cases, including both unilateral and bilateral forms, reveal a numerical preponderance of horizontal hemianopia affecting the lower half of the visual field, unilateral involvement being much more frequent than bilateral involvement. Well-defined bilateral horizontal hemianopia of the upper half of the visual

field is extremely rare. Of the 35 cases of horizontal hemianopia which *Zanen and Brihaye* (1948) found in the literature, 8 involved the upper and 27 the lower half of the visual field. The 6 patients of *di Marzio, Gavina and de Nigris*, who were included among these 35 cases, comprised 5 cases of horizontal hemianopia affecting the lower half of the visual field, and 1 case in which the upper half was affected; in both of the bilateral cases, the visual defects were situated in the lower halves. The 15 cases of bilateral horizontal hemianopia, affecting either the upper or lower halves, which *Willbrand and Sängner* had recorded up to the year 1915, i.e. 6 cases of *hemianopsia superior* and 9 of *hemianopsia inferior*, probably give a misleading impression of the relative incidence of these two forms. As for the type and localisation of the lesions responsible for horizontal hemianopia, here it is difficult to produce statistical evidence, because there are several factors involved which render such evidence of dubious value:

(a) The number of patients reported on is too small to be of much statistical significance. Moreover, since they consist of very heterogeneous cases, often described on the basis of single observations and of unsatisfactory diagnoses, it is difficult to apply any uniform principles to their evaluation.

(b) In many cases the diagnosis is arachnoiditis affecting the optic chiasma and the intracranial segments of the optic nerve – a diagnosis which is unsatisfactory and even controversial. *Zanen and Brihaye* encountered this diagnosis in 10 of their 35 cases; in another paper, by *Magin A. Diez* (1955), this same diagnosis appears 9 times among a total of 24 cases.

(c) A large proportion of cases of bilateral horizontal hemianopia are attributable to war wounds (transverse gun-shot wounds through the occiput). A review which were to include such cases would be distorted and would not reflect the “natural” frequency distribution with respect to aetiology and localisation.

There is one point on which all the studies available agree, i.e. that only in exceptional cases does a tumour of the brain give rise to horizontal hemianopia. This is borne out most clearly by the investigations of *Tönnis* (1955), who studied the ophthalmological symptoms presented by 3033 patients suffering from brain tumours: among the 849 patients (= 34.5 %) with visual-field defects, unilateral horizontal hemianopia was found only in 2 cases; the bilateral form was not encountered at all.

In the following discussion, we shall attempt to present as complete

a review as possible of horizontal hemianopia affecting the upper half of the visual field; however, insofar as it may be of interest to differentiate this condition from, or contrast it with, horizontal hemianopia affecting the lower half of the field, we shall also refer, where necessary, to the latter as well.

II. Detailed Analysis

(1) Possible Causes of Horizontal Hemianopia Occurring along the Visual Pathway, with Particular Reference to Hemianopsia Horizontalis Superior

(a) Among the *intra-ocular* causes are those in which the horizontal hemianopia is, as it were, a logical anatomical consequence of the damage sustained, and others in which it constitutes an incidental complication.

Included in the first group are *occlusions of the main upper or lower branch of the central retinal artery*. According to *Walsh*, the boundary between the unaffected and the blind half of the visual field runs horizontally through the fixation point; according to *Lauber*, the dividing line is either sharp or slightly undulated (this being referred to as a distinctive characteristic of angioscotomas, in contrast to pure neuroscotomas of the retina). *Dubois-Poulsen* has described as follows a special form of demarcation line which he considers typical of this pathogenesis: on the nasal side there is a sharp dividing line along the horizontal meridian, whereas on the temporal side the sighted half overlaps the horizontal line in a shallow arc; in between, the blind spot bulges out into the remaining half of the visual field. This pattern was at one time regarded as proof of a "vascular raphe overlying the neural raphe" (*Traquair*), but studies on injection preparations have since revealed clear evidence of capillary intercommunications between the ramifications of both the nasal and the temporal retinal arterioles (*François*). This peculiarity in the demarcation line can be explained by reference not only to the architecture of the retinal fibres, but also to the fact that the extent of a visual-field defect of vascular origin is dependent on two factors: firstly, on the size of the area of damage sustained by the ganglion cells and, secondly, on the pattern of their nervous fibres in relation to the ischaemic zone of the retina.

Where occlusion of one of the main branches has occurred, central

visual acuity may still be completely unimpaired (*Dubois-Poulsen, Walsh*). Occasionally, the reason for the preservation or "sparing" of a macular field of vision is found to be a cilioretinal artery. *Lauber* has reported cases in which the visual defect was so sharply bounded that the patient scored 6/6 when the eye was tested, although only the lower or upper half of the optotype was visible. The occurrence of symmetrical defects in both eyes must probably be regarded as a freak finding; however, a case of bilateral horizontal hemianopia affecting the upper half of the visual field, and due to occlusion of both main inferior branches of the retinal artery, has been reported by *Traquair* (quoted in *Franceschetti*). In all cases where there are visual defects due to vascular occlusion, it must be borne in mind that the form of the scotomas may vary: this failure to conform to a standard pattern may be ascribed to gradations in the damage present – i.e. in cases of only partial occlusion – and to individual peculiarities in the architecture of the retinal vessels.

According to *Dubois-Poulsen*, *glaucoma* is a very common cause of horizontal hemianopia; *Walsh*, too, refers to *glaucoma* as a causative factor and mentions the possibility of a sharp dividing line between the functioning and nonfunctioning portion of the visual field, either in one eye or simultaneously in both eyes.

The cause is presumably disparate pressure-induced damage to fibres and vessels in the upper and lower regions of the retina. Such damage may initially manifest itself in the form of *Rönne's* nasal step in one of the nasal quadrants; later it may give rise to an expanding arcuate scotoma or may lead to a progressive narrowing of the periphery until one complete half of the visual field is lost.

In cases of horizontal hemianopia which have developed in this way, it must be added, however, that owing to the temporal raphe the boundary is only well defined on the nasal side and that concentric contraction of the remaining half of the visual field can be expected to occur.

As a rule, *Rönne's* nasal step originates in the upper nasal quadrant, indicating that it is the inferior arcuate fibres which are the first to sustain damage. This is consistent with the observations of *Proksch* (1927), who found that, in 100 patients with defects of the visual field due to *glaucoma*, the upper half was more damaged than the lower half in 62 cases, the lower half more damaged than the upper in 20 cases, and both halves more or less equally damaged in 18 cases. In *glaucoma*, therefore, we can expect – in contrast to the normal rule –

to find horizontal visual-field defects more frequently in the upper than in the lower half.

It is a well-known fact that *severe haemorrhages* may be followed by visual disorders ranging from scotomas to complete and permanent blindness. The site of these usually recurrent haemorrhages is almost always the digestive system or the female genital tract; the visual disturbance sets in either as an immediate haemorrhagic complication or after a lapse of 3 to 7 days. Generally both eyes are affected, and the condition may be permanent or only temporary, in which case the visual disorder will either disappear completely or merely result in mild residual impairment of vision. The form of the defect in the visual field varies considerably (hemianopias, scotomas, restrictions in the field of vision); in the literature, however, there are repeated references to the fairly regular occurrence of horizontal hemianopia, almost invariably affecting the lower half of the visual field.

Dubois-Poulsen describes the horizontal dividing line as irregular; but, on repeated occasions, he observed a strong resemblance to the dividing line which he considers to be typical of occlusion of one of the main branches of the central retinal artery. On the other hand, in one case reported by *Franceschetti* (1949) and in another reported by *Uthoff* (1922), the line of division was exactly horizontal. As a rule, central vision is impaired in such cases, although it may be completely unaffected (*Dollfus, Franceschetti*).

Loss of vision in the upper halves of the visual fields is extremely rare. *Franceschetti* refers to several older reports of such cases (*Manz*, 1873; *Landesberg*, 1877; *Naegeli*, 1879 and *Grout*, 1914); but the most impressive example is a case of bilateral horizontal hemianopia affecting the upper halves, which *Dollfus* (1951) found in a patient with haematemesia caused by a gastric ulcer. Incidentally, in this case the horizontal boundary conformed fairly closely to the pattern as specified by *Dubois-Poulsen*.

Regarding the pathogenesis of post-haemorrhagic visual disorders, the hypothesis frequently advanced at one time was that they were due to retinal ischaemia. The rather variable ophthalmoscopic findings seemed to offer no evidence to dispute this theory. Inspection of the eyegrounds may reveal thin arterioles, haemorrhages, retinal oedema, and a swollen papilla of pale or glassy appearance. In the case of visual-field defects extending downwards, it was assumed that haemorrhagic hypotension and the force of gravity acting upon the

blood probably increase the risk of an impaired blood supply to the upper portions of the retina.

Piper and Unger (1957) offer a different explanation. Having investigated the comparatively regular occurrence of *hemianopsia horizontalis inferior* in temporal arteritis and as a sequel to haemorrhages, they point out that in both of these conditions, the pathological-anatomical substrate of the visual disorders is an anaemic infarct in the optic nerve, situated just behind the lamina cribrosa and above the central retinal artery. They postulate that this infarct affects the terminal ramifications of the anterior branch of the nutritive *a. centralis nervi optici*, which – running above the central retinal artery as it does – supplies precisely those fibres that stem from the upper portions of the retina.

Also mentioned as further causes of horizontal hemianopia are *retinal detachment* and various other *retinopathies* – although in such cases the condition tends to be more in the nature of a chance occurrence and the most that one can expect to find are pictures approximating to those of horizontal visual-field defects. Much the same applies to an observation reported by *Streiff and Hermann* (1950), an observation which nevertheless deserves special attention in view of its diagnostic significance: it concerns two cases of frank bilateral impairment of vision in the upper halves of the visual field; some time later (in one case after 7 years, and in the other after one year) both patients presented an ophthalmoscopic picture of pigmental degeneration of the retina.

(b) Only very sparse data are available on damage to the *optic nerve* as a cause of *hemianopsia horizontalis superior*. In *syphilitic atrophy of the optic nerve*, it is possible for both upper or lower halves of the visual field to be lost (*Walsh*) – a loss which as a rule, however, is associated with contraction of the remaining half of the visual field, the boundary running only roughly along the horizontal line. In contrast to this is an example quoted by *Wilbrand and Saenger*, in which there was tabetic atrophy of the optic nerve with quite pronounced bilateral horizontal hemianopia of the upper half, but completely unimpaired vision in the remainder of the visual field.

In addition to this purely infectious aetiology, physical influences are also liable to enter into play. It is possible, for instance, that a *fracture of the bony opticnerve canal* may affect one of the upper halves of the visual field. According to *Walsh*, the same type of visual defect – but bilateral – can be expected in *oxycephaly*; here, he suggests

that stretching of the optic nerves and pressure on their lower portions are possibly the chief pathogenetic factors. Mechanical damage may also result from *sclerosis, atheromatous plaques, tortuosities and aneurysms* of neighbouring blood vessels. The vessels most likely to be implicated are the ophthalmic artery, the carotid artery, and the anterior cerebral artery (*Liebrecht*). It must be realised that the lesion may be attributable either to the direct effect of pressure or to the *contrecoup* phenomenon. Among a group of patients selected as illustrative of this arteriosclerotic-mechanical pathogenesis, *Knapp* (1932, 1940) cites two occurrences of horizontal hemianopia, one being a case of incomplete binocular hemianopia affecting the lower half of the visual field, and the other a perfect example of binocular hemianopia of the upper half; in both instances, sclerosis of the carotid arteries was confirmed by x-ray examination. In such cases, however, one can never be certain as to the role played by anaemic infarcts due to similar sclerotic lesions of the nutrient vessels supplying the optic nerves. Where the onset and progression of a visual-field defect follows a regular and consistent pattern, this might well be indicative of damage from without.

In the presence of an aneurysm it is easier to accept the mechanical theory. In view of its topographical relationships with the optic nerve, the ophthalmic artery deserves particular attention in this connection in cases where horizontal visual-field defects have developed. Although aneurysms of this artery are extremely rare, two cases have in fact been reported: one case of unilateral, incomplete hemianopia affecting the upper half of the visual field (*del Martel, François, Guillaume*) and another in which not one, but both eyes were involved, owing to the presence of aneurysms in each of the two ophthalmic arteries (*Sandford, Craig and Wagener*, quoted in *Duke-Elder*).

The fact that certain types of *pre-sellar and parasellar tumours* may in exceptional cases lead to horizontal hemianopia by exerting pressure on the intracranial segment of the optic nerve, raises the question of damage to the optic chiasma – a problem which will be discussed later.

Finally, attention should be drawn to one rather curious observation reported by *Harrington*. This author found a broad *coloboma of the optic nerve* as the anatomical substrate of a well-defined superior hemianopia.

References to further fascicular causes have only been found in connection with defects of the lower halves of the visual field. These

comprise: appropriately situated *neuritis of the optic nerves* (*Wilbrand and Saenger, Magin A. Diez*) which is also known to produce fairly symmetrical and exact bilateral forms (*Walsh*), *disorders affecting the blood supply to the fasciculi optici* (already mentioned with reference to temporal arteritis), and another rarity reported by *Harrington*, i.e. bilateral hemianopia of the lower halves of the visual field in two (treated) cases of *pernicious anaemia*; in both patients the clinical picture was complicated by additional neurological disorders.

(c) If we ascend further up the visual pathway and examine the possible connections between the *optic chiasma* and horizontal hemianopia, we must first of all note the close topographical and pathological-functional links existing between the region where the two optic nerves cross and the bilateral *pars intracranialis nervi optici*. According to *Traquair*, it is possible, for example, that if the chiasma is pushed upwards by a subchiasmal tumour, the optic nerves may be pressed against the vessels of the *circulus arteriosus Willisi* or against the dural fold at the upper margin of the optic-nerve canal; this may result not only in chiasmal symptoms, but also in unilateral or bilateral visual-field defects of the type found in horizontal hemianopia of the lower half. Indeed, in most cases of horizontal field defects due to involvement of the chiasma, it is difficult to proceed on the assumption that the lesion is located solely in the chiasma and to exclude the possibility of additional damage to the intracranial segments of the optic nerves.

If, in the light of present-day knowledge concerning the course of the fibres in the chiasma, we try to postulate a pathological process which would of necessity produce a horizontal visual-field defect, it is in the anterior portion of the chiasma, immediately contiguous to the optic nerves, that such a process is most likely to occur. It is in this area, where the retinal projection is still roughly the same as in the *fasciculi optici*, that pressure from above or below could conceivably lead to a horizontal defect.

The question as to what types of damage in the region of the chiasma are capable of producing such visual-field defects, and how often this diagnosis is made in cases of horizontal hemianopia, can be answered on the basis of the literature as follows:

A *tumorous aetiology* is rare. Tumours which attack the chiasma from below (suprasellar meningiomas, pituitary tumours) give rise only in exceptional cases to horizontal hemianopia (*Hartman, Huber*). Proof – albeit not quite conclusive – that horizontal hemianopia may

occur in this way, is to be found in a case reported by *Praun and Pröscher* (1900). The patient, a woman presenting a clinical picture of acromegaly, lost her vision in both upper halves of the visual field over a period of months. The characteristic visual defect caused by subchiasmal tumours (the more exactly median the site of the tumour, the more marked the loss of vision) is, however, a form of bitemporal hemianopia beginning in the upper temporal quadrants.

Pressure on the chiasmal region originating from above (tumours of the third ventricle, internal hydrocephalus) is also stated in the literature to be only a very rare cause of horizontal visual-field defects (*Huber, Magin A. Diez*).

Cushing (quoted in *Walsh*) has observed a type of autochthonous chiasmal process in the shape of gliomas which resulted in horizontal hemianopia.

Finally, the site of origin of pre-sellar and parasellar tumours, which have been referred to in connection with horizontal defects of the visual field, is such that they come into contact primarily with the intracranial segments of the optic nerves or, at the utmost, with the anterior portions of the chiasma; significantly enough, horizontal hemianopia constitutes only one of the stages occurring in progressive impairment of the visual field.

Tumours of this kind include, for example, meningiomas which grow in a strictly medial direction on the lesser wing of the sphenoid bone connected with one of the anterior clinoid processes. The visual-field defect resulting from damage to the intracranial *fasciculus opticus* begins as a rule in the upper nasal quadrant, but quite often it subsequently extends over the mid-line and develops into horizontal hemianopia of the upper field. Later, the chiasma may also become involved (*Duke-Elder*). The same pathogenesis is to be found in two cases reported by *Kendree and Doshay*: in one of them, a neurinoma of the left olfactory tract had led to transitory hemianopia of the lower field in the right eye, and in the other case a meningioma of the lesser wing of the sphenoid bone was accompanied by horizontal hemianopia of the upper field in the left eye and incipient hemianopia of the same type in the right eye.

In the reviews of cases of horizontal hemianopia to which reference has already been made (*Zanen and Brihaye, Willbrand and Saenger*), chiasmal involvement is relatively the most frequent cause adduced; but, at the same time, the diagnosis of *opticochiasmatic arachnoiditis* which was made in almost all these cases is undoubtedly the least

satisfactory. *Zanen and Brihaye*, who have undertaken a critical analysis of this pathogenesis, reach the following conclusions:

(1) It is difficult in principle to explain a horizontal hemianopia by reference to the course of the fibres in the chiasma.

(2) In arachnoiditis there is usually no convincing anatomical connection between the visual-field defect and the implicated noxa.

(3) Only in exceptional cases has freeing of the adhesions by surgical means resulted in any improvement.

These authors conclude that in the majority of cases arachnoiditis is not, in fact, the cause and that the origin of the horizontal hemianopia probably lies in the pre-chiasmal area; they consider that damage of the type we have already discussed, i.e. damage due to pressure exerted by blood vessels, is the most likely explanation.

Before leaving the region in which the optic nerves cross, and before ascending further along the visual pathway, we should like at this point to make just a brief reference to the diagnostic significance of atrophy of the optic nerve. It is evident, firstly, that unilateral optic atrophy is involved in all forms of pre-chiasmal damage to the visual pathway which result in the destruction of neural substance; secondly, that lesions located between the chiasma and the primary optical centres give rise to gangliopetal atrophy of both optic discs; and, thirdly, that where damage has been sustained above the geniculate bodies, due diagnostic significance must be ascribed to the absence of paling.

(d) The *optic tract* coincides with the beginning of that portion of the visual pathway damage to which typically results in an homonymous defect in both visual fields on the side contralateral to the lesion. Consequently, a monolateral horizontal hemianopia is *a priori* inconceivable where there is a unilateral lesion above the chiasma; the only possibility, in fact, is that the bilateral form may occur owing to the presence together of two homonymous upper or lower quadrant anopias on opposite sides.

This condition has never been reported in cases of damage to the optic tract, and its occurrence is indeed highly unlikely. In the first place, damage confined solely to the optic tract is in itself rare; secondly, in cases of partial interruption, such as would be necessary to produce horizontal hemianopia, incongruent visual-field defects are so common that they even serve as clues to the localisation of the pathological process. In this connection, we would remind readers of our earlier remarks on the architecture of the optic-tract fibres.

This is a convenient point at which to add a few further comments on the topical diagnosis of retrochiasmal lesions of the visual pathway. Of major importance is the question as to which segments of the pathway are most likely to be involved in cases where the visual-field defect constitutes only a partial symptom of a complex neurological picture. From the relevant topographical features it can be deduced that a maximum of neighbourhood symptoms will probably be encountered where the segment involved is that running from the optic tract to the *carrefour sensitif*. Outside this zone the incidence of neighbourhood symptoms diminishes, so that occipital lesions, for instance, may well produce visual-field defects unaccompanied by any further complications.

It is well-known that hemianopic pupillary paralysis serves as a very useful differential-diagnostic sign in the presence of a lesion in the optic tract or in the optic radiation – the absence of such paralysis being indicative of damage above the lateral geniculate body; but it is also an equally well-known fact that this symptom is difficult to detect.

“Sparing of the macula” in cases of hemianopia is a problem which cannot be discussed here. Suffice it to say that there are two opposing schools of thought on this subject: according to one of them, this macular sparing has a certain amount of diagnostic value, inasmuch as its presence suggests damage to the optic radiation, although its absence permits of no topical diagnosis (Huber, 1950); the opposing view is that this phenomenon provides no clue whatsoever to the location of the lesion (Sachsenweger, 1963).

(e) From what we now know about the cyto-architectural features in this area, it would appear that, in the *lateral geniculate bodies*, the upper quadrants of the homonymous hemiretinae are represented in a medial segment, and the two lower quadrants in a lateral segment. To produce a horizontal hemianopia of the upper visual field, the damage must accordingly affect the lateral side of both geniculate bodies, whereas damage to the medial side will result in horizontal hemianopia of the lower visual field.

In view of the fact that a lesion in this segment of the visual pathway is only rarely diagnosed, it is fortunate that records exist of a case in which this theory of the pathogenesis of horizontal hemianopia was confirmed at autopsy. Here, inspection of the brain revealed lesions concentrated chiefly on the external aspects of the geniculate

bodies and caused by syphilitic vascular changes. That the horizontal hemianopia – affecting the upper half of the visual field – was only of a partial nature is merely of quantitative significance and in no way invalidates the congruence between the clinical picture, the local findings, and the theory accounting for the pathogenesis of the hemianopia. The authors in question also examined the vascularisation of the affected area and found that branches of the anterior cerebral artery from within, and branches of the anterior choroid artery from without, extended to the geniculate bodies (*Mackenzie, Meighan, Pollock*, 1933). It is therefore conceivable that a defect in one of these vascular beds, due to a selective lesion in a geniculate body, may lead to a more or less regular quadrantic anopia.

Finally, reference should be made to yet another hypothesis advanced by *Walsh*. According to *Walsh*, preservation of central visual acuity in the presence of horizontal hemianopia affecting either the upper or the lower half of the visual field is characteristic of trypanosamide poisoning; he believes the cause to be vascular disorders in the region of the geniculate bodies. In man, however, no such finding has yet been discovered at autopsy; in animal experiments, on the other hand, haemorrhages have been found in the optic tract (in monkeys) and in the optic chiasma (in rabbits), and – as reported by *Grant* – also atrophy affecting the ganglionic layer of the retina.

(f) The *optic radiation* and the *visual cortex* offer the most favourable terrain for the development of horizontal hemianopias, because here there is a marked spatial segregation between the areas of representation corresponding to the upper and lower portions of the retina. The broad vertical sheet of fibres in the optic radiation follows the curvature formed by the outer wall of the posterior horn of the lateral ventricle; consequently, the ventricle interposes itself to some extent between the upper zone of fibres, belonging to the superior homonymous quadrants of the retina, and the lower zone corresponding to the inferior quadrants. The extensive intermediary segment is occupied by the binocular-macular bundle. In the striate area the homonymous hemiretinae are divided horizontally by the calcarine fissure, the upper lip of which corresponds to the superior quadrants, and the lower lip to the inferior quadrants. Following each other in a fronto-occipital direction, and in their own specific locations, are the demilune (which is only monocular), the extramacular zones, and the very large area of representation of the macula. As regards the finer structure of these tissues, the immediate proximity of fibres or

cells which project identical points from the retina is a phenomenon which gradually becomes more and more pronounced in the course of the optic radiation, with the result that even minor homonymous defects display a marked degree of congruence.

Bearing in mind these anatomical relationships, we can expect to find horizontal hemianopias showing a perfectly horizontal dividing line. Central vision may be either spared or reduced, depending on the degree to which the damage extends into the macular area.

Since we are concerned here chiefly with horizontal hemianopia affecting the upper half of the visual field, we must deal first of all with *vascular causes* – not because these are the commonest, but because they are the most likely to be encountered in this particular instance. The possibility of horizontal defects begins to arise at approximately the middle third of the broad vertical sheet of fibres, i.e. in the region where the middle cerebral artery supplies mainly those segments corresponding to the upper retinal quadrants and where the posterior cerebral artery supplies chiefly the areas of representation for the lower quadrants. A distinctive feature of the macular zone of the cortex is the fact that it is supplied with blood from both of these arteries. Bilateral ischaemia in the respective vascular beds of the posterior cerebral artery may therefore result in damage to both inferior halves of the optic radiation or to both lower lips of the calcarine fissure, which in turn will lead to horizontal hemianopia affecting the upper half of the visual field. The fact that encephalomalacia of both occipital lobes is a strikingly common autopsy finding, coupled with experimental evidence indicating that the occipital lobes are somewhat susceptible to bilateral involvement (*Best*), also points to the possibility of vascular damage as a pathogenetic factor.

Among patients exemplifying the classic pathogenesis of bilateral horizontal hemianopia, i.e. destruction of the appurtenant areas of grey or white matter by transverse *gun-shot wounds in the occipital region*, horizontal hemianopias of the upper field have been found only in exceptional cases (e.g. *Best*, 1917). Owing to the close topographical affinities with vital structures of the brainstem and the transverse sinus, a bullet wound through the lower lips of the calcarine fissure usually proves fatal, whereas injury to the upper lips is quite compatible with life.

Other forms of cranial trauma which may give rise to horizontal visual-field defects include *fractures* and *haemorrhages*; one example is a case reported by *Berkley and Bussey* (1950), in which a cerebellar

haemorrhage had caused damage to both upper lips of the calcarine fissure.

Regarding the aetiological role played by *tumours*, *Huber* states that horizontal hemianopias have also been observed in the presence of tumours involving the occipital portion of the interhemispheric fissure.

Finally, mention should also be made of a possible functional cause, i.e. *ophthalmic migraine*, which may produce visual-field defects sometimes in the upper halves and sometimes in the lower halves.

(2) *Personal Observations in a Case of Hemianopsia Horizontalis Superior Bilateralis*

(a) Extracts from the case history:

Patient: Emil H.-H., born 1888.

Family history: no data worthy of note.

Personal anamnesis: in 1946 and 1958 a centrally translucent patch of infiltration was observed in the left lung, as well as pleural thickening; T.B.-negative. 1957 – suspected of having sustained a posterior-wall infarction, atrial flutter. 1961 – mild diabetes of late onset diagnosed. Chronic alcoholism.

No knowledge of previous eye diseases; last reliable data: 1954, vision in both eyes 6/5.

Admitted to hospital on 15. 6. 1961. Ocular symptoms, in the form of transient blurring of vision, had appeared about three months previously; the ophthalmologist consulted had discovered no pathological findings.

Frank visual disturbances (in the meantime the patient had been able to read again) did not set in until 23. 5. 1961, after which there was a rapid deterioration in vision (no information available on the visual field) accompanied by severe headaches. According to the patient and his wife, there were no symptoms of paresis, but since the onset of the visual disorders he had become “very forgetful”.

Ophthalmological status at the time of admission to hospital: Vision R 0.3; L finger-counting at 3 metres; no improvement with spectacles.

Projection of light from below correctly registered by both eyes; but patient uncertain when light projected from above.

Purkinje's figure visible on both sides; no difference between upper and lower half.

Reaction of the pupils sluggish on both sides, but otherwise normal.

Corneal sensitivity on both sides 2 gr/mm²; intra-ocular pressure left and right 16 mm Hg.

No impairment of bulbar motility.

Colour vision: patient able to distinguish between blue and yellow, but red and green both appear grey and can hardly be differentiated.

Morphological status (findings identical on both sides):

Anterior segments of the eyeball normal, apart from senile changes. Eyegrounds: optic disc of normal colour and configuration, but entire disc somewhat cupped.

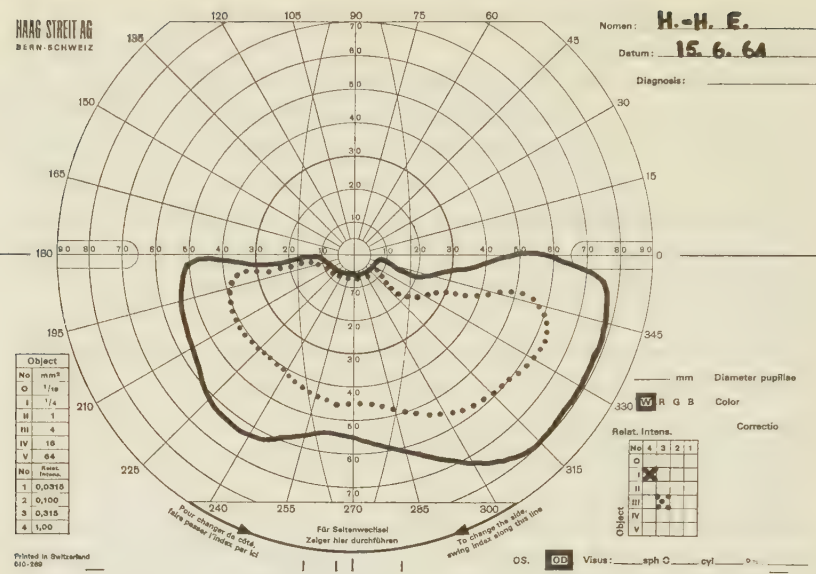


Fig. 1

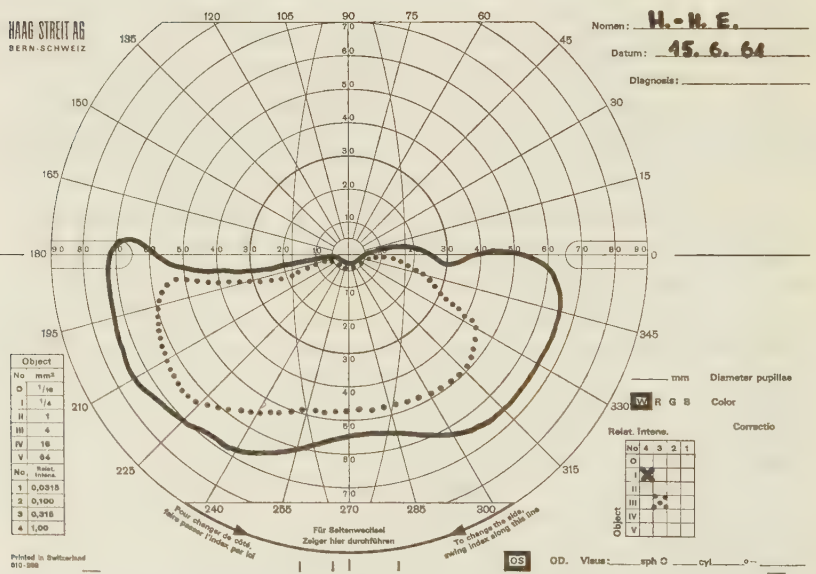


Fig. 2

Sclerosis of the retinal vessels. Slight pigmentation shifts in the centre of the retina, but no macular degeneration as such.

Visual fields: horizontal hemianopia affecting the upper half of the visual field; normal range of vision in the lower halves; no macular sparing (Figs. 1 and 2).

Neurologist's report (from the University Neurological Polyclinic, Basle): Examination was difficult, owing to the presence of a psycho-organic syndrome. The neurological examination provided no reliable clues to the localisation and aetiology of the visual-field defect. The anamnesis also failed to yield any evidence of neurological disorders.

The internal specialist who examined the patient (at the University Medical Polyclinic, Basle) reported the following findings: Generalised arteriosclerosis associated with a mild psycho-organic syndrome; diabetes of late onset; pulmonary emphysema; bilateral pleural thickening, with some calcification, and possibly a pleural effusion on the left side.

On the basis of these pulmonary findings, which showed a marked deterioration as compared with the findings in 1958, the specialist was unable definitely to exclude the possibility of a tumour.

Unfortunately it was not possible to tap the pleural effusion because, although such treatment was certainly indicated, the patient insisted on returning home before the examinations had been completed.

During the patient's stay in hospital, the following changes occurred:

(1) An extension of the isopters (Mark III/3 – Goldmann perimeter), but not beyond the horizontal meridian. This was presumably attributable to the fact that the patient's eyes had become accustomed to the examination procedure. In all other respects both fields of vision showed no changes whatsoever, despite repeated examination (Figs. 3 and 4).

(2) Vision in the left eye improved to 0.1.

(b) *Epicrisis*. This patient showed the rarest type of horizontal hemianopia, i.e. hemianopia characterised by loss of vision in both upper halves of the visual field. An attempt to clarify the aetiology of the condition can be made by approaching the problem from three angles:

Firstly, by reference to the form and development of the hemianopia.

The fact that the visual defects were congruous, that the course of the dividing line was exactly horizontal, and that the residual visual fields were of normal extent led us to suppose that the visual pathway had probably been interrupted in an area where the horizontal division is anatomically represented and where the requisite conditions for exact homonymous defects prevail. The rapidity with which

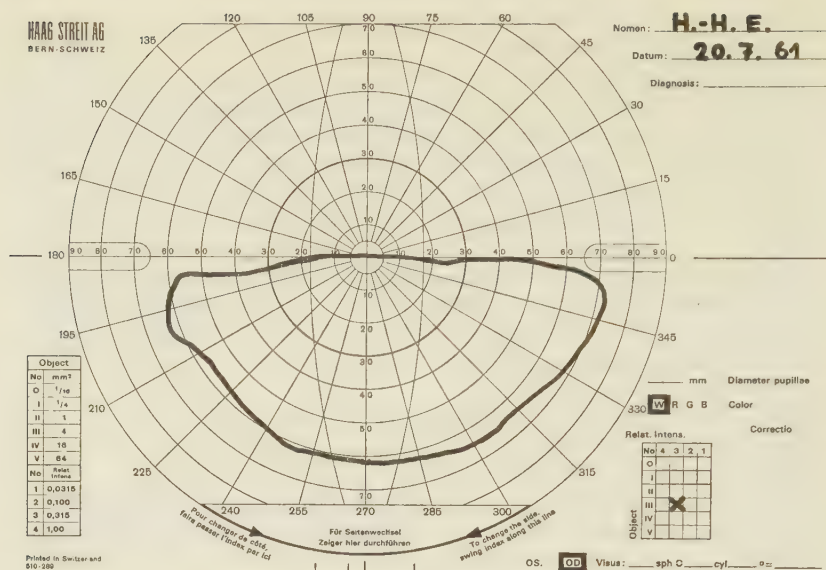


Fig. 3

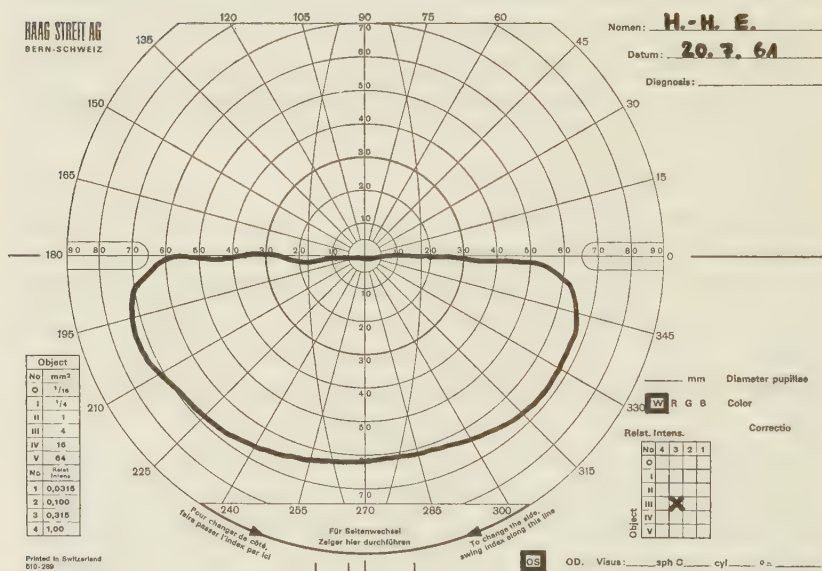


Fig. 4

the visual disorder developed was suggestive of a vascular process. Taken together, these two items of evidence pointed to symmetrically located encephalomalacic lesions in the region supplied by the posterior cerebral artery – lesions which had resulted in damage either to the two lower halves of the broad vertical sheets of fibres in the optic radiation or to both upper lips of the calcarine fissure. That the dually vascularised area of macular representation had also been put out of action, could be accounted for either by some deviation from the norm or by senile vascular impoverishment of the cerebral cortex due to the presence of arteriosclerosis.

Secondly, by reference to other ophthalmological or neurological symptoms.

The possibility of an intra-ocular cause, or of a cause located in the optic nerve, could be excluded. There were no neurological neighbourhood symptoms whatsoever. The patient merely showed a psycho-organic syndrome, the progressive nature of which had been noted by others during the period prior to his admission to hospital. The absence of accompanying neurological signs might also be regarded as indicative of an occipital process.

Thirdly, by reference to previous experience concerning the incidence of bilateral horizontal hemianopia affecting the upper half of the visual field.

Here, again, the patient's clinical picture left us with virtually no choice other than to assume a vascular cause located in the occipital lobe.

Unfortunately, we were unable to clarify the pulmonary findings, which might have shed light on the possibility of metastases in the occipital lobes; it is highly unlikely, however, that two tumour metastases would have resulted in such strikingly congruent visual-field defects.

Summary

The term horizontal or altitudinal hemianopia is defined, and the clinical picture differentiated from other forms of hemianopia.

After a discussion on the incidence of horizontal hemianopia, with reference to forms affecting the upper half of the visual field, the lower half, and either one eye or both eyes, a review – based on a study of the literature – is given of all those types of damage to the visual pathway in which such visual-field defects occur either as a typical sequel or as an incidental finding. At the same time an attempt is

made to establish, with respect to each segment of the visual pathway, a connection between the noxa, the anatomical substrate, and the nature of the resultant visual-field defect.

Finally, personal observations in a case of horizontal hemianopia are discussed: the patient was suffering from the rarest form of horizontal visual-field defect, i.e. bilateral horizontal hemianopia affecting the upper half of the field, the dividing line running exactly along the horizontal meridian, below which the inferior halves of the visual field were of perfectly normal extent. The cause was presumed to be a bilateral vascular disorder of the visual pathway located in the area where the middle cerebral artery supplies chiefly the upper half of the optic radiation as well as the upper lip of the calcarine fissure and where the posterior cerebral artery (in the vascular bed of which the disorder must in this case have been situated) is responsible for nutrition of the corresponding lower parts.

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